IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re to application of: P. BRINGMANN et al.
Serial No.: 10/005,646
Filed: 7 December 2001
For: Novel Fibroblast Growth Factors
Art Unit: 1647
Examiner: Christine J. Saoud

DECLARATION

I BRANKA MITROVIC declare and state as follows:

I am currently a Senior Scientist in the Department of Immunology, Berlex Biosciences, United States (the American subsidiary of Schering AG, Germany). In 1982, I received my B.S. (Bachelor of Science degree) in biochemistry and microbiology, from the School of Science ("Mihailo Pupin"), University of Belgrade, Belgrade. In 1989, I received my M.D. (Medical Doctor degree) from the School of Medicine, University of Belgrade, Belgrade.

Since receiving my M.D., I have had extensive experience in medical and biological research in the laboratories of various major academic institutes and pharmaceutical companies as detailed in the attached copy of my curriculum vitae. As a Senior Scientist at Berlex Biosciences, I direct and supervise multiple research programs for identifying novel therapeutics targeting Central Nervous System (CNS) regeneration, Spinal Cord Injury (SCI), and other CNS diseases (OND). I am also a named inventor on the above-referenced patent application.

Overall, I have 17 years of research experience in the fields of neurobiology and glial biology, and 11 years of experience in pharmacology developing therapeutics for the treatment of Multiple Sclerosis (MS) and other neurological diseases such as Parkinson's Disease (PD), Amyotrophic Lateral Sclerosis (ALS), and Spinal Cord Injury (SPI). Specific interests of my research include oligodendrocyte biology, demyelination/remyelination, axonal regeneration, and the role of growth factors particularly in the induction of CNS regeneration, as indicated by my list of publications in the attached curriculum vitae.

With respect to the Examiner's comments concerning what is taught by Webster et al. and Nakamura et al., I am providing the following remarks which support the belief

that the findings of Webster et al. together with the findings of Nakamura et al. do not teach or suggest the use of FGF-9 for the treatment of multiple sclerosis (MS). In contrast to the Examiner's belief, the findings of Webster et al. indicate that the biological activities of fibroblast growth factors differ and that some of these biological activities would not be beneficial for the treatment of MS; and the findings of Nakamura et al. do not suggest or support the use of FGF-9 for the treatment of MS and report that FGF-9 promotes proliferation of astrocytes, which as explained below would have an adverse and undesired impact for the treatment of MS.

Webster et al.

The Examiner states that "Webster teaches that growth factors, including FGFs, are involved in proliferation, differentiation and survival of cells in the oligodendroglial lineage, and that oligodendrocytes are the cells that form the myelin sheaths. Webster teaches that administration of growth factors could increase proliferation of progenitor oligodedrocytes, enhance their differentiation, upregulate the synthesis of myelin constituents and promote myelin regeneration in the adult CNS, which would be beneficial for treatment of MS."

However, Webster et al. actually state that "FGF-2 treatment of mature oligodendroglia expressing myelin specific proteins failed to induce mitosis and led to the cell death by apoptosis" (see page 116, column 2, last full para., sentence 4). The same was also reported in vivo by Goddard et al. (2001) Mol. Cell Neurosci. 2001 18(5):557-69 ("Fibroblast growth factor-2 inhibits myelin production by oligodendrocytes in vivo").

Therefore, the teachings of Webster et al. actually indicate that fibroblast growth factors such as FGF-2 that act on and promote the proliferation of astro-glia progenitors would <u>not</u> be beneficial for the treatment of MS. For the same reasons detailed below, the teachings of Nakamura et al. referencing the findings of Naruo et al. ((1993) J. Biol. Chem. 268:2857-2864) for the effect of FGF-9 on promoting the proliferation of astrocytes and astro-glia progenitors, do not teach or suggest the use of FGF-9 for the induction of remyelination in MS.

Nakamura et al.

The Examiner states that Nakamura et al. "teach a number of biological activities for FGF-9, including the ability to promote proliferation of primary cortical astrocytes, oligodendrocyte type 2 astrocyte progenitor cells fibroblasts and neuron like PC-12 cells". However, Nakamura et al. did not investigate the biological activities of FGF-9.

Instead, Nakamura et al. investigated only the localization of FGF-9 protein and mRNA expression in the rat central nervous system by immunohistochemistry and in situ hybridization and state the following.

- With respect to FGF-9 protein: "In addition to the neuronal localization of FGF-9 immunoreactivity that we reported previously, the present double-label immunohistochemistry clearly demonstrated that the immunoreactivity was present in glial fibrillary acidic protein (GFAP)-positive astrocytes preferentially present in the white matter of spinal cord and brainstem of adult rats and in CNPase-positive oligodendrocytes that were arranged between the fasciculi of nerve fibers in cerebellar white matter and corpus callosum of both adult and young rats. There was a tendency for FGF-9 immunoreactivity in oligodendrocytes to be more pronounced in young rats than in adult rats. The variation of oligodendrocyte FGF-9 immunoreactivity in adult rats was also more pronounced than that in young rats. "
- With respect to FGF-9 mRNA: "With in situ hybridization, FGF-9 mRNA was observed in astrocytes in the white matter of rat spinal cord and oligodendrocytes in the white matter of cerebellum and corpus callosum of adult and young rats. The expression of FGF-9 mRNA in glial cells was lower than in neurons, and not all glial cells expressed FGF-9. In the present study, we demonstrated that FGF-9 was expressed not only in neurons but also in glial cells in the CNS."

Therefore, Nakamura et al. do not provide any teaching regarding the biological activities of FGF-9 on myelinating cells. However, Nakamura et al. references Naruo et al. (1993) concerning the effect of FGF-9 on different cells when stating that FGF-9, promotes "proliferation of primary cortical astrocytes, oligodendrocyte type 2 astrocyte progenitor cells fibroblasts and neuron like PC-12 cells" (see page 54, column 1, sentence 5). However, astrocyte proliferation is not a desired feature of a potential MS therapeutic. Astrogliosis (hypertrophy and proliferation of astrocytes) is one of a major pathological features of MS and, more particularly, astrogliosis is considered responsible for the failure of myelin regeneration and axonal repair in MS lesions. (e.g., see Malik et al. (1998) J. Neurolimmunol. 86(2):155-62 ; and Fawcett et al. (1999) Brain Res. Bull. 49(6):377-91).

In addition, a proposed mechanism of IFN-beta efficacy in the treatment of MS is the ability of the drug to reduce astrocytosis and thereby promote endogenous repair (Malik et al., 1998). Contrary to the teachings of Nakamura et al. and consistent with the teachings of the present application, I and the co-inventors in the present application,

have demonstrated that FGF-9 inhibits, and not enhances, the proliferation of primary rat cortical astrocytes.

In addition consistent with the teachings of the present application, the present inventors have also demonstrated that FGF-9 stimulates the proliferation of oligodendrocytes (OLs), and not "oligodendrocyte type 2 astrocyte progenitor" as taught by (Nakamura et al. in referencing Naruo et al.) and as stated by the Examiner. This distinction is important, since "oligodendrocyte type 2 astrocyte progenitors" are not committed cells and can become either astrocytes or oligodendrocyte progenitors. Again, the induction of proliferation of astrocyte progenitors or astrocytes is not considered desirable effect for the treatment of MS. Such findings, consistent with the teachings of the present application, were recently confirmed by Fortin et al. (2005) J. Neurosci. 25(32):7470-9, who have reported that "FGF-9 specifically targets "differentiated OLs". In addition, Fortin et al. report that FGF-2, through the activation of FGFR1, leads to the proliferation of oligodendrocyte progenitors, and produces deleterious effects on differentiated OLs (i.e., aberrant reentry into cell cycle and down-regulation of myelin proteins with a loss of myelin membrane), while FGF-9 does not. Therefore, the differences between the activity of FGF-9 on mature oligodendrocytes versus "oligodendrocyte type 2 astrocyte progenitors" is an important difference with respect to the regulation of both normal OL development and potential OL/myelin repair.

The Present Specification

Examiner also states that "the instant specification basis the claimed invention on the ability of FGF-9 to stimulate PC-12 cells , which are obtained from adrenal gland. Therefore, based on the biological activity of FGF-9 on "cells of neuronal origin , Applicant asserts that FGF-9 would be useful for treatment of MS. This is exactly what is taught in prior art, etc." I believe the Examiner has misinterpreted or taken out of context the teachings and claims of the application when stating that the present specification basis the claimed invention "on the ability of FGF-9 to stimulate PC-12 cells". Importantly, for clarification, the present inventors have demonstrated the following:

1. FGF-9 induces the proliferation and survival of the cells of committed oligodendrocyte lineage and which is important in the induction of remyelination in MS patients (e.g., see Figure 5 of the present application); and
2. FGF-9 has the activity on primary rat cortical neurons (e.g., see Figure 9 of the present application).

In view of the results provided in the present specification, an expert in the field would reasonably expect that FGF-9 and/or FGF-20 would induce myelin formation and remyelination in vivo and, consequently, be useful for the treatment of disease involving demyelination or the failure of remyelination as claimed in the patent application. The studies described in the present specification are directed to therapies that stimulate oligodendrocyte proliferation and survival, and the development of therapeutics that can induce remyelination and restoration of the function in a disease involving demyelination or a failure to remyelinate, such as MS. Remyelination allows the return of saltatory conduction and the functional recovery of demyelination-induced deficits. However, remyelination is often incomplete in the adult human central nervous system, and this failure of remyelination is one of the main reasons for clinical deficits in demyelinating diseases such as MS. Further, prior studies in the field report that the failure of remyelination in MS is contributed to by a depletion of oligodendrocytes, especially following repeated episodes of demyelination (see e.g., review by Keristead, H.S. et al. (1999) 468:183-197 Adv. Exp. Med. Biol.). Thus, in view of the approach and results described in the present specification, one would reasonably expect that stimulating oligodendrocyte proliferation and survival would result in the induction of remyelination in vivo and treatment of the disease.

As demonstrated by the in vitro studies provided in the patent application, FGF-9 and/or FGF-20 induce the proliferation of oligodendrocytes. Further, I and the co-inventors of the present application have demonstrated that both FGF-9 and/or FGF-20 are capable of inducing the expansion of oligodendrocytes in vivo and subsequent remyelination. Specifically, we have demonstrated in vivo that FGF-9 administered systemically, also induces expansion and myelination of SVZ cells transplanted into myelin deficient mice (shi/shi -/-).

Therefore, in view of the teachings of the present application, an expert in the field would reasonably expect that FGF-9 and/or FGF-20 would be effective in stimulating oligodendrocyte proliferation, survival, and myelination in vivo as confirmed by the present inventors, and be useful as a therapeutic for inducing remyelination and restoration of the function in a patient having a disease involving demyelination or a failure to remyelinate, such as MS.

I hereby declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true; and

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further that these statements were made with the knowledge that willful false statements may jeopardize the validity of the patent application or any patent issued thereon.

Dated: 5/25/06 By: Branka Mitrovic

Branka Mitrovic, M.D.

Curriculum Vitae of Branka Mitrovic, MD

PROFESSIONAL EXPERIENCE

Berlex Biosciences, Schering AG, Department of Immunology
Senior Scientist- 2005- current

- Direct and supervise the research programs aimed to identify novel therapeutics targeting CNS regeneration in Multiple Sclerosis, spinal cord injury and ONDs
- Project Team Leader for **"FGF-9 for CNS Repair"** – *B0 Project*
- Core Team member of **"Spheramine for the treatment of PD"** IPT
- Project Team Member of **"RPTPsigma inhibition for CNS regeneration"** project
- Initiated **"VGSC1.6 inhibition for axonal regeneration"** project
- Initiated **"Mesopram for SCI regeneration"** project
- Initiated **"GPR-19 for CNS regeneration"** project
- Coordinate the work with Gene Therapy Department in order to establish the GT Technology Platform for the Viral Based Delivery in to CNS
- Supervised the collaboration between SAG and Victoria Neurosciences- Australia – Berlex representative in SAG/NSV Working Group
- Co-Project Leader for **"Identification of novel genes involved in the differentiation of precursor cells into oligodendrocytes "**- *NSV-04*

Scientist III- 1999 – 2004

- Direct and supervise the research programs aimed to identify novel therapeutics for treatment of Remyelination in Multiple Sclerosis
- Project Team Leader for **"Novel CNS Specific FGFs and their role in CNS Repair"** – *C0 Project*
- Project Team Leader for **"Biogenic Estriol-Diester Prodrugs for the Treatment of Multiple Sclerosis"**- *B0 Project*- member of IDT
- Project Team Member for **"KLK-6 as a Potential Target for the MS Treatment"**
- Coordinate the work with Gene Therapy Department in order to establish the GT Technology Platform for the Viral Based Delivery in to CNS – technologies evaluated VLP, AAV and HSV
- Project Team Member for **"CNS Specific GT delivery vectors"** –*C0 Project*
- Initiated **"IGF-I-R modulators and CNS Regeneration"** and **"Anti-TNF-R1 Approach for the Treatment of Multiple Sclerosis"** Project
- Provided intellectual and technical support to the **"Spheramine for the treatment of PD"** Project
- Supervised the collaboration between SAG and Victoria Neurosciences- Australia – Berlex representative in SAG/NSV Working Group
- Co-Project Leader for **"Identification of novel genes involved in the differentiation of precursor cells into oligodendrocytes "**- *NSV-04*
- Establish joint BBS/ NSV/RCJ programs especially the joint SAG program in the "New target identification".
- Manage the interface between Immunology, BBS and Neurology, SAG Departments and coordinate the activities on the joint Projects such as: Microglia Activation Inhibitors, Fyn-kinase inhibitors, Peroxinitrate decomposition Catalysts, and Neuroimmunophilins
- Coordinate the work between Immunology, BBS and Ginecology & Andrology, SAG Departments and coordinate the activities on the **ER-b agonist Project**
- Coordinated the activities between the Immunology and Animal Pharmacology Department in order to establish demyelinating animal models of MS such as LL induced Demyelination in rats as well as animal models of Neurodegeneration- carotid artery occlusion (CAO) in gerbils
- Initiated genomic programs aimed toward identifying the novel therapeutic targets from murine, rat and primate models of Multiple Sclerosis
- Supervised collaboration with outside investigator to acquire tissue from MS patients and primate animal models of demyelination
- **Evaluated all external MS / CNS related** programs such as: IFNb enhancer (Transition Therapeutics), IFNtau (Pepgen), Mynocyclin derivatives (Paratek Pharmaceuticals), XIFNg mAb (ABI), Albuferon (HGS), Mitoxantron derivatives (ABR), Neliximab (GenPath), MBP8298 (BioMS), PN-277 (Proneuron), Fipamezole (Juvantia), APL (Neurokrine), C11AG (BioSphyngs), HE 2200,2500 (Hollis-

Eden), Talampanel (IVAX) as well as programs and technologies from Centaur, AXIS- Selera, ZYCOS, Cogent, Via Cell, Cephalon, NeuronZ, Celltics, Amrad, Receptron, UCSF, UCI, UCLA
- Organized CNS Regeneration Advisory Board.
- Member of Berlex' Scientific Advisory Committee
- Member of GRC Committee
- Member of CNS Regeneration Working Group
- Published Book Chapter on the MS treatment
- Supervise scientists, associated scientists and technical specialists (direct supervisor)
- Invited and hosted outside scientists as consultants and seminar speakers
- Organize Workshops on the anti-inflammatory and reparative approaches for the treatment of Multiple Sclerosis and Stroke – "The Fight against Stroke- Strategies for the Treatment of Brain Ischemia" (2001); "Multiple Sclerosis –New Strategies for the Treatment of Autoimmune Demyelination" (2002); and ESRF Workshop "Opportunities and Challenges of the Therapies Targeting CNS Regeneration" (2004)

Berlex Biosciences, Schering AG, Department of Immunology
Scientist II- 1995- 1999
- Perform original research on demyelination and remyelination in Multiple Sclerosis
- Project Team leader for **"Mesopram for Remyelination"** – *C0 Project*
- Determine mechanism of action of Mesopram as it relates to MS- Work published
- Design strategies and establish protocols and experimental designs leading to discovery of novel therapeutics (small molecules and novel growth factors) targeting remyelination and repair in CNS
- Initiated collaboration with Bioinformatics group in order to identify CNS specific growth factors by using genomics approaches – searching Incyte and other databases using HMM and SS models
- Project Team member for **"SST Remyelination project"**
- Established bioassays for the proliferation, differentiation and migration of the cells of neuronal and glial origin
- Established reporter gene assays for the modulation of MBP gene expression
- Set up immunological cell based assays measuring the expansion, migration and differentiation of primary cells and cell lines of lymphocytic, mononuclear and astrocytic origin
- Investigate the role of different PDE isoform inhibitors on the induction of neurite outgrowth
- Developed *in vitro* myelinating system for testing the effects of novel therapeutics on the formation of compact myelin
- Design strategies for testing the potential therapeutics in animal models of MS, stroke, PD and other neurodegenerative diseases
- Established outside collaborations for studying brain availability of potential therapeutics
- Provide intellectual and technical expertise to the research programs addressing the role of Chemokines in MS including CCR-8 Project
- Investigated the role of novel Interferon on the inhibition of gliosis
- Provided support to the other Project Teams: p60, Lipoxins, MIM30, TREM-2, MDL-1
- Evaluated the role of IGF-I, IGF-I / BP3 in the *in vivo* induction of Remyelination
- Manage the collaboration between Immunology, BBS and Neurology, SAG Departments and coordinate the activities on the joint Projects such as: Microglia Inhibitors, AMPA antagonists, and Neuroimmunophilins
- Invited and hosted outside scientists as consultants and seminar speakers

Department of Neurology, School of Medicine, University of California, Los Angeles, 1992-1994
NIH Fogarty Research Scientist
- Conducted research on mechanism of demyelination in Multiple Sclerosis and CNS AIDS.
- Outstanding UCLA Postdoctoral Scholar Award recipient.
- NIH Fogarty International AIDS Research Fellowship recipient
- Acquired funding through the grant from UCLA Program in Psychoneuroimmunology.
- Determined The Role of Nitric Oxide in Glial Pathology
- Initiated and directed studies identifying Nitric Oxide as a Potential Pathologic Mechanism in Demyelination- Work Published
- Investigated the Differential Effects of NO on Primary Glial Cells *In Vitro*.- Work Published
- Determined the mechanism of action of NO mediated toxicity as it relates to MS- Work Published

- Established an *in vitro* Model of Oligodendrocyte Destruction by Nitric Oxide - Work Published
- Identified the differential involvement of glial subtypes in iNOS expression and NO production- Work Published
- Described developmentally regulated sensitivity of oligodendrocytes to NO – Work Published
- Published invited review articles
- Reviewed scientific papers for *Brain, Behavior and Immunity* and *Developmental Neuroscience* journals
- Extensive experience in the characterization of immunologic and CNS-specific cells types; analysis and quantification of RNA and protein expression including in situ hybridization and ICH plus standard molecular biological and immunologic techniques
- Responsible for and directed a group of post-doctoral fellows, graduate students, and technicians

School of Public Health, University of California, Los Angeles, 1991-1992
Postdoctoral Research Fellow
- Performed studies on gene expression and spatial distribution of various brain antioxidant enzymes (GPx, SOD, GR, catalase)- Work published
- Established various molecular and cell biology protocols and procedures in the laboratory

Department of Pathology, School of Medicine, University of California, Los Angeles, 1990-1991
Postdoctoral Scholar
- Carried out research on mechanism of neurotoxicity caused by MeHg and other organometals
- Presented work at national and international scientific conferences

EDUCATION
- **Medical Doctor**, 1989, School of Medicine, University of Belgrade, Belgrade
- **Bachelor of Science** in Biochemistry and Microbiology, 1982, University of Belgrade, Belgrade

ACADEMIC HONORS AND AWARDS
- Outstanding UCLA Postdoctoral Scholar Award recipient
- National Academy of Arts and Sciences Fellowship recipient
- Academic Excellence Award and City of Belgrade Student Fellowship recipient

RESEARCH GRANTS AND FELLOWSHIPS
- NIH Fogarty International AIDS Research Fellowship recipient.
- UCLA Program in Psychoneuroimmunology Grant

PROFESSIONAL TRAINING
- Advanced Course in Immunology- AAI accredited
- Drug Development Process Training
- Project Management Training (PM for Research, Team Effectiveness, Negotiation and Problem Solving for PM)
- Global Institute for Leadership Development (GILD) Training

PATENTS
1. Doms R, Edinger AL, **Mitrovic** B, Zhou Y, Faulds D, Collman RG, Hesselgesser J, Horuk R, Doms R "Methods and composition for modulating the interaction between the APJ receptor and the HIV virus" – Patent No: US 6,475,718 B2, Nov. 5, 2002

2. Bringman P, Faulds D, **Mitrovic B**, Srinivisan S,. "Novel fibroblast growth factors" PCT WO 02/46424 A2 (13.06.2002)

PUBLICATIONS

1. Terayama R, Bando Y, Jiang YP, **Mitrovic B**, Yoshida S. Differential expression of protease M/neurosin in oligodendrocytes and their progenitors in an animal model of multiple sclerosis. Neurosci Lett. 2005 Jul 1-8;382(1-2):82-7. Epub 2005 Apr 11

2. **Mitrovic B**, Stock G, Perez HD, Dinter H; "Molecular aspects of MS treatment" in Molecular Medizin, Band 5, Erkrankungen des Zentralnervensystems, 2001

3. Dinter H, Tse J, Halks-Miller M, Asarnow D, Onuffer J, Faulds D, **Mitrovic B**, Kirsch G, Laurent H, Esperling P, Seidelmann D, Ottow E, Schneider H, Tuohy VK, Wachtel H, Perez HD. The type IV phosphodiesterase specific inhibitor mesopram inhibits experimental autoimmune encephalomyelitis in rodents. *J Neuroimmunol.* 2000 Aug 1;108(1-2):136-46.

4. Edinger AL, Hoffman TL, Sharron M, Lee B, Yi Y, Choe W, Kolson DL, **Mitrovic B**, Zhou Y, Faulds D, Collman RG, Hesselgesser J, Horuk R, Doms RW. An orphan seven-transmembrane domain receptor expressed widely in the brain functions as a coreceptor for human immunodeficiency virus type 1 and simian immunodeficiency virus. *J Virol.* 1998 Oct;72(10):7934-40.

5. **Mitrovic B**, Stock G, Perez HD; "Multiple sclerosis: from rugs to riches" *J. Mol. Med.* 75,75 (1997)

6. Parkinson J, **Mitrovic B**, Merrill JE; "The Role of Nitric Oxide in Multiple Sclerosis." *J. Mol. Med.* 75, 174 (1997)

7. Merrill JE, Murphy SP, **Mitrovic B**, Mackenzie-Graham A, Dopp JC, Ding M, Griscavage J, Ignarro LJ, Lowenstein CJ; "Inducible Nitric Oxide Synthase and Nitric Oxide Production by Oligodendrocytes." *J. Neurosci. Res.* 48,1 (1997)

8. **Mitrovic B**, Parkinson J, Merrill JE; "An *in vitro* Model of Oligodendrocyte Destruction by Nitric Oxide and Its Relevance to Multiple Sclerosis." In Methods, A Companion to *Methods in Enzymology*, 10, 501 (1996)

9. **Mitrovic B**, Ignaro LJ, Vinters HV, Akers M-A, Schmidt I, Uittenbogaart C, Merrill JE; "Nitric Oxide Induces Necrotic But Not Apoptotic Cell Death in Oligodendrocytes." *Neurosci.* 65 (2), 531 (1995)

10. **Mitrovic B**, St. Pierre BA, Mckenzie-Graham AJ, Merrill JE; "The Role of Nitric Oxide in Glial Pathology" *Annals New York Acad. Sci.* 738, 436 (1994)

11. Mackenzie-Graham AJ, **Mitrovic B**, Smoll A, Merrill JE; "Differential Sensitivity to Nitric Oxide in Immortalized, Cloned Murine Oligodendrocyte Cell Lines " *Dev. Neurosci.* 16, 162 (1994)

12. **Mitrovic B**, Martin FC, Ignaro LJ, Anton PA, Shanahan F, Merrill JE; "Neurotransmitters and Cytokines in CNS Pathology." *Prog. Brain Res.* 103, 319 (1994)

13. **Mitrovic B**, Ignarro LJ, Montestruque S, Smoll A, Merrill JE; "Nitric Oxide as a Potential Pathologic Mechanism in Demyelination: Its Differential Effects on Primary Glial Cells *In Vitro*." *Neurosci.* 61 (3), 575 (1994)

14. Buckman TD, Suthpin MS, **Mitrovic B**; "Oxidative stress in a clonal cell line of neuronal origin: Effects of antioxidant enzyme modulation." *J. Neurochem.* 60 (6), 2024 (1993)

MEMBERSHIPS

American Association of Immunologists
American Society for Cell Biology
New York Academy of Sciences